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Assessment of atrioventricular conduction in aortic valve disease[1]

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SUMMARY To determine the frequency of atrioventricular conduction disturbances in aortic valve disease, 26 consecutive patients (age 54 ± 2 years) with symptomatic aortic valve disease were studied by His bundle electrocardiography at the time of cardiac catheterisation and were compared with a group of patients who underwent cardiac catheterisation and were found to have coronary artery or mitral valve disease but no aortic valve disease. Patients with aortic valve disease had significantly longer PR, AH, and HV intervals than cardiac patients not having this abnormality. Patients with aortic stenosis had prolonged HV, 52 ± 6 vs 42 ± 2 ms ($P = 0.06$), whereas patients with chronic aortic regurgitation had prolonged PR, 245 ± 27 vs 163 ± 5 ms ($P < 0.001$), and prolonged AH, 178 ± 30 vs 102 ± ms ($P < 0.001$). Patients with combined lesions had significant prolongation of PR, AH, and HV intervals. Three patients with acute aortic regurgitation caused by endocarditis had normal atrioventricular conduction. Though the presence of valvular calcification did not significantly alter the pattern of atrioventricular conduction in these patients, those with calcified aortic valves had longer HV ($P < 0.005$) than the control group. In addition, ventricular dysfunction or coronary artery disease did not affect the pattern of atrioventricular conduction in these patients. Thus, atrioventricular conduction disturbances are common in symptomatic aortic valve disease. With aortic stenosis the site of delay occurs more frequently below the His deflection, whereas in aortic regurgitation it is more frequent above the His deflection.

Occurrence of heart block in calcific aortic valvular disease has been recognised since the earliest clinical-pathological studies of patients with Adams-Stokes disease (Stokes, 1846; Yater and Cornell, 1935; Warshawsky and Abramson, 1947; Harris *et al.*, 1969). The proximity of the aortic valve and its supporting structures to the atrioventricular conduction system could explain this association (Lev, 1964). His bundle electrocardiography is a useful technique for localising the site of atrioventricular block and, in some instances, can show atrioventricular conduction abnormalities even before they are apparent on the surface electrocardiogram (Damato *et al.*, 1969). Though patients with calcific aortic valve disease have been included in previous His bundle studies done on patients with atrioventricular block (Bharati *et al.*, 1974; Rosen *et al.*, 1975) there has been no His bundle study of patients with aortic valve disease *per se*. Accordingly, it is the purpose of this study to show the His bundle electrocardiographic findings in patients with aortic valve disease. The results of this study, which are reported here, show that atrioventricular conduction abnormalities are common in symptomatic aortic valve disease.

Method

The study group consisted of 26 patients undergoing diagnostic cardiac catheterisation for symptomatic aortic valve disease. The control group was obtained from 25 consecutive patients undergoing diagnostic cardiac catheterisation and found to have significant organic heart disease but no evidence of aortic valve disease. Aetiological diagnoses of both groups are shown in Tables 1 and 2. Patients having given informed consent were studied at the time of diag-

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Table 1 *Diagnosis in patients studied*

Control (N = 25)	*Aortic valve disease (N = 26)*
Coronary artery disease (23)	Rheumatic (8)
Rheumatic mitral stenosis (1)	Congenital bicuspid (6)
Congenital atrial septal defect and mitral prolapse (1)	Endocarditis (4)
	Syphilis (1)
	Calcific aortic sclerosis (5)
	Unknown (2)

nostic cardiac catheterisation before any contrast studies. All patients were postabsorptive. None had received cardiac antiarrhythmic medication for at least one week before cardiac catheterisation, except for three patients with acute infective endocarditis, who were studied at the time of emergency cardiac catheterisation.

His bundle electrograms were recorded using a modification of the standard technique (Scherlag *et al.*, 1969). Electrocardiograms from standard leads with filters set at a frequency response of 1 to 200 Hz and His bundle electrograms with filters set at a frequency response of 40 to 500 Hz were recorded simultaneously on a multichannel, oscilloscopic photographic recorder (Electronics for Medicine DR-12) at a paper speed of 100 mm/s. A bipolar electrode catheter was introduced percutaneously into the right femoral vein and passed across the tricuspid valve. It was gradually withdrawn until a characteristic His deflection was observed at a time when A and V were both prominent. The interval between the A and V was carefully explored to identify any split His deflections. Deflections occurring 31 ms or less before the V were assumed to represent the right bundle-branch deflection. The AH interval was measured from the onset of the first high frequency component of the A to the onset of the H deflection. HV interval was measured from the onset of the H deflection to the onset of ventricular depolarisation on a standard electrocardiographic lead or the ventricular electrogram, whichever occurred earlier. PR interval was measured from the onset of the P wave to the onset of the R wave on the standard limb lead that had the longest duration. The intervals obtained represented the average of 10 cycles. Patients with sinus bradycardia had atrial pacing performed and corrected sinus recovery times were measured, using the methods of Narula and co-workers (1972).

Haemodynamic measurements were made using standard methods. Mean systolic gradient across the

Table 2 *Clinical and haemodynamic data*

	Case no.	*Age (y)*	*Diagnosis*	*Gradient (mmHg)*	*Cal*	*AR*	*CI (l/min per m²)*	*EF (%)*	*Cor*
(A) Aortic stenosis (n = 6)	1	57	Bicuspid	67	Yes	+	3·8	89	LAD Rt
	2	65	Tricuspid calcific	44	Yes	+	—	39	LAD
	3	55	Tricuspid calcific	67	Yes	+	2·9	51	Patent
	4	57	Rheumatic	52	Yes	+	3·2	—	ND
	5	69	Tricuspid calcific	20	Yes	None	3·0	67	ND
	6	59	Bicuspid	80	Yes	None	—	53	ND
Mean ± SE		60 ± 2		55 ± 9			3·2 ± 0·2	60 ± 9	
(B) Aortic regurgitation, chronic (n = 9)	7	38	Rheumatic	None	None	3+	1·5	—	ND
	8	53	Unknown	None	None	3+	3·0	60	Patent
	9	51	Rheumatic	None	None	2+	3·2	63	ND
	10	30	Bicuspid	None	None	3+	3·0	55	ND
	11	47	Unknown	None	None	3+	3·0	43	ND
	12	68	Syphilis	None	None	4+	2·5	39	ND
	13	37	Rheumatic	None	None	4+	2·8	—	ND
	14	56	Rheumatic	None	Yes	3+	2·2	46	LAD CX
	15	60	Endocarditis	None	None	3+	2·3	—	Patent
Mean ± SE		49 ± 4					2·6 ± 0·2	51 ± 4	
(C) Aortic stenosis and regurgitation (n = 8)	16	48	Rheumatic	59	Yes	3+	3·0	64	Patent
	17	62	Bicuspid	35	Yes	3+	4·0	73	Patent
	18	60	Rheumatic	80	Yes	3+	2·5	68	Patent
	19	56	Bicuspid	65	Yes	3+	2·9	—	ND
	20	57	Rheumatic	55	Yes	3+	3·6	65	Patent
	21	62	Tricuspid calcific	35	Yes	3+	3·5	59	Patent
	22	57	Bicuspid	60	Yes	3+	ND	—	ND
	23	65	Tricuspid calcific	66	Yes	3+	2·2	42	Patent
Mean ± SE		58 ± 2		56 ± 5			3·3 ± 0·2	62 ± 4	
(D) Aortic regurgitation, acute (n = 3)	24	55	Endocarditis	None	None	4+	3·4	56	ND
	25	43	Endocarditis	None	None	3+	4·0	—	ND
	26	47	Endocarditis	None	None	4+	2·1	40	ND
Mean ± SE		48 ± 4					3·9 ± 6	48 ± 8	

Abbreviations: EF, Ejection fraction; ND, not done; CX, circumflex coronary artery stenosis; Rt, right coronary artery stenosis; Cal, valvular calcification; Cor, coronary arteriography; LAD, left anterior descending artery stenosis; AR, aortic regurgitation; CI, cardiac index.

Table 3 *Electrophysiological data*

	Case no.	*Electrocardiogram*	*HR/min*	*PR (ms)*	*AH (ms)*	*HV (ms)*
(A) Aortic stenosis (n = 6)	1	LAE, NAST-T	85	180	100	47
	2	NAT	65	160	95	40
	3	LVH, LAE, NAST-T	90	190	120	42
	4	LVH, IVCD, LAE, NAST-T	90	200	120	70
	5	LVH, NAST-T	90	160	80	70
	6	LVH, NAST-T	84	165	110	45
Mean ± SE			84 ± 5	176 ± 7	104 ± 6	52 ± 6
(B) Aortic regurgitation, chronic (n = 9)	7	LBBB, LAE	85	440	390	50
	8	LVH, AbnLAD	80	280	220	50
			86	270	220	50
			63	330	270	50
	9	LVH, LAE	65	240	190	50
	10	LVH, NAST-T	70	180	130	35
	11	LVH, AbnLAD, NAST-T	71	200	100	45
	12	LBBB, LAE	88	285	200	55
	13	LVH, NAST-T	77	200	124	43
	14	LVH, NAST-T	78	200	110	81
	15	LVH, LAE, NAST-T	85	210	140	40
Mean ± SE			77 ± 3	248 ± 27	178 ± 30	48 ± 4
(C) Aortic stenosis and regurgitation (n = 8)	16	LVH, IVCD, LAE, NAST-T	90	220	133	51
	17	LVH, LAE	70	230	170	49
	18	LAE, NAST-T	68	220	150	70
	19	LVH, LAE	80	210	140	55
	20	LVH, LAE, SSS, NAST-T	60	240	138	79
	21	LVH, LAE, SSS, NAST-T	56	161	104	36
	22	LVH, LAE, NAST-T	72	200	120	55
	23	LVH, LAE, NAST-T	71	200	144	35
Mean ± SE			71 ± 4	211 ± 9	137 ± 7	53 ± 5
(D) Aortic regurgitation, acute (n = 3)	24	LAE, AbnLAD, NAT	55	140	60	45
	25	Normal	43	180	90	50
	26	LAE, NAST-T, AbnLAD	47	190	95	50
Mean ± SE			48 ± 4	170 ± 15	81 ± 11	48 ± 2

Abbreviations: NAST-T, nonspecific abnormality of ST segment and T waves; LBBB, left bundle-branch block pattern; AbnLAD, abnormal left axis deviation; NAT, non-specific abnormality of T wave; LVH, left ventricular hypertrophy; SSS, sick sinus syndrome; LAE, left atrial enlargement; IVCD, intraventricular conduction defect.

Table 4 *Summary of haemodynamic and electrophysiological data in control and aortic valve disease patients*

	Control (n = 25)	*Aortic valve disease (n = 26)*	*P value*
Age (y)	52 ± 2	54 ± 2	NS
CI (l/min per m²)	2·8 ± 0·1	3·1 ± 0·1	NS
EF (%)	57 ± 5	56 ± 3	NS
HR/min	76 ± 4	81 ± 2	NS
PR (ms)	163 ± 5	211 ± 11	< 0·001
AH (ms)	102 ± 5	137 ± 12	< 0·025
HV (ms)	42 ± 2	50 ± 3	< 0·025

P value indicates level of significance when comparing groups.
Abbreviations: See Tables 2 and 3.

aortic valve was determined by a left ventricular-aortic withdrawal tracing; cardiac output was measured by the indicator dilution technique (Zierler, 1962), using indocyanine green, and ejection fraction by the area-length method (Dodge *et al.*, 1966), using a single plane 30° right anterior oblique projection. The degree of aortic regurgitation was estimated from the opacification of the left ventricle after aortography using a scale of 0 to 4+ with 1+ indicating incomplete lining of the left

Table 5 *Summary of haemodynamic and electrophysiological data in patients with chronic aortic valve disease*

(A) *Aortic stenosis (n = 6)*		
Age (y)	60 ± 2	P < 0·05
CI (l/min per m²)	3·2 ± 0·2	NS
EF (%)	60 ± 9	NS
HR/min	84 ± 5	NS
PR (ms)	176 ± 7	NS
AH (ms)	104 ± 6	NS
HV (s)	52 ± 6	NS (P = 0·06)
(B) *Aortic regurgitation (n = 9)*		
Age (y)	49 ± 4	NS
CI (l/min per m²)	2·6 ± 0·2	NS
EF (%)	51 ± 4	NS
HR/min	77 ± 3	NS
PR (ms)	248 ± 27	P < 0·001
AH (ms)	178 ± 30	P < 0·005
HV (ms)	48 ± 4	NS
(C) *Aortic stenosis and regurgitation (n = 8)*		
Age (y)	58 ± 2	NS
CI (l/min per m²)	3·3 ± 0·2	NS
EF (%)	62 ± 4	NS
HR/min	71 ± 4	NS
PR (ms)	211 ± 9	P < 0·001
AH (ms)	137 ± 7	P < 0·005
HV (ms)	53 ± 5	P < 0·005

P value indicates level of significance when values shown were compared to those of control group.
Abbreviations: See Tables 2 and 3.

ventricle and 4+ indicating better opacification of the left ventricle than the aorta after 2 to 3 beats. Differences between groups studied were compared using Student's t test for unpaired data. Values shown are the means ± one standard error of the mean.

Results

The electrocardiographic findings of the patients with aortic valve disease are shown in Table 3. When the age, cardiac index, ejection fraction, and heart rate of patients with aortic valve disease were compared with the control group no difference was found. Patients with aortic valve disease, however, had longer PR, AH, and HV intervals (Table 4). Six patients had isolated aortic stenosis with an average aortic valvular mean systolic gradient of 55 mmHg. These patients were older than the control group but had normal cardiac function (Table 5). PR and AH intervals were not longer than those of the control group. HV was longer, though only of borderline significance, P = 0·06 (Table 5).

Nine patients had chronic aortic regurgitation and no aortic systolic valvular gradient. Their age, cardiac index, ejection fraction, and heart rate were not different from the control group. However, average PR interval was 248 ± 27 ms and AH interval was 178 ± 30 ms in these patients, both longer than in patients not having aortic valvular disease (Table 5). His bundle tracing obtained from one patient with aortic regurgitation is shown in Fig. 1. The AH interval is prolonged and a non-conducted A is shown with block above the His deflection. Six months after aortic valve replacement, this patient was admitted with atrial flutter; cardioversion by rapid atrial pacing was performed. The AH interval after cardioversion was 220 ms. One year later he was admitted with dizziness and was found to have advanced atrioventricular block, with block occurring above the His deflection. A permanent pacemaker was inserted at that time. Another patient, with a very prolonged AH interval of 390 ms before operation, was found to have periods of Wenckebach-type atrioventricular block after operation when he was not receiving digitalis. On the other hand, one patient with calcific aortic regurgitation and no aortic stenosis was found to have a prolonged HV, measuring 85 ms and normal AH (Fig. 2).

Eight patients had combined aortic regurgitation and aortic stenosis; they had an average aortic valvular mean systolic gradient of 56 mmHg and 3+ aortic regurgitation. While their average age, cardiac index, ejection fraction, and heart rate were not different from control patients, these patients had prolonged PR, AH, and HV intervals (Table 5). Four patients had previous infective endocarditis. Three, who were studied less than four months after its occurrence, had normal AV conduction despite being on digitalis. One patient, who was studied three and a half years afterwards, had a prolonged AH interval. Coronary arteriography was performed on 13 patients, all of whom had angina pectoris. Three of these patients had significant coronary artery disease and only one of these patients had abnormal atrioventricular conduction. Moreover, the control group was made up largely of patients with coronary artery disease; therefore, the abnormalities found in the patients with aortic valve disease could not be explained by the presence of associated coronary artery disease. When the cardiac function was com-

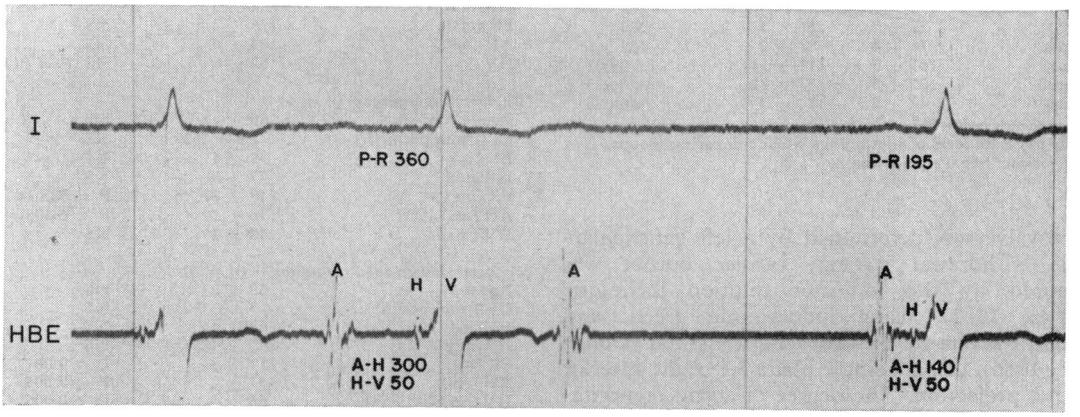

Fig. 1 *Recording of electrocardiogram (lead I) and His bundle electrogram (HBE) in a patient with aortic regurgitation showing prolongation of AH and nonconducted A. Paper speed at 100 mm/s. Interval between time lines 1 s.*

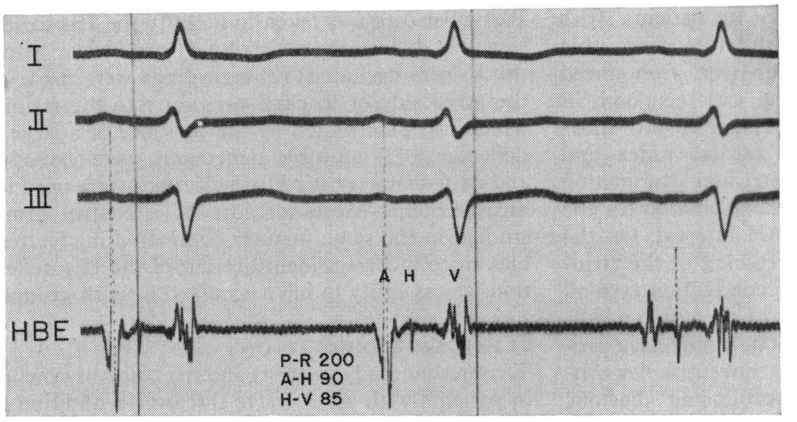

Fig. 2 *Recording of electrocardiogram (leads I, II, and III) and His bundle electrogram (HBE) in a patient with calcific aortic regurgitation showing prolongation of HV interval. Paper speed at 100 mm/s. Interval between time lines 1 s.*

pared, patients with aortic valve disease and normal cardiac performance (cardiac index equal to or greater than 2·5 l/m per m^2 and ejection fraction equal to or greater than 50%) did not have a pattern of atrioventricular conduction different from those with depressed left ventricular function (a cardiac index less than 2·5 l/m per m^2 or an ejection fraction of less than 50%). Patients with chronic valve disease were also analysed to see whether or not aortic valvular calcification was present. Though patients with valvular calcium had a longer HV interval than those not having this finding, 55 ± 4 vs 46 ± 2 ms, this difference was not significant. When compared with the control group, however, patients with calcified aortic valvular apparatus had longer HV conduction ($P < 0.005$). In addition, 2 patients with combined aortic stenosis and regurgitation who had sinus bradycardia were found to have prolonged sinus recovery times. One of these patients had prolonged AH and HV intervals while the other had normal atrioventricular conduction.

Discussion

The present study shows that atrioventricular conduction disturbances are common in chronic, symptomatic aortic valvular disease. More than 50 per cent of the patients in the present study had atrioventricular nodal and/or His-Purkinje conduction times outside the normal range found in the control group. The sensitivity of His bundle electrocardiography in revealing disturbed atrioventricular conduction in patients with aortic valve disease is also shown in this study. Four patients had prolonged AH and/or HV intervals when their surface electrocardiogram had a normal PR interval. Three of these patients had an HV interval of 70 ms or more. Moreover, the utility of His bundle electrocardiography is particularly well illustrated in this study, since surface lead determinations of atrioventricular conduction were taken as the *longest* PR interval obtained, whereas intracardiac determinations were taken as the *shortest* measurements of atrioventricular conduction.

In view of the proximity of the His bundle to the aortic valve (Lev, 1964), the finding of HV prolongation in patients with calcific aortic stenosis and/or regurgitation is not unexpected. Previous clinical-pathological studies have shown calcific, fibrotic lesions invading or in close proximity to the cardiac conduction system in some patients with atrioventricular block (Stokes, 1846; Yater and Cornell, 1935; Warshawsky and Abramson, 1947; Harris *et al.*, 1969). On the other hand, a dissociation of electrophysiological and necropsy anatomical findings has been reported in a patient with calcific aortic stenosis and transient heart block (Rosen *et al.*, 1975). The failure to observe HV delay in some patients with pronounced calcification of the aortic valvular apparatus might be a reflection of such a discordance of electrophysiological and anatomical findings. Furthermore, since the determination of aortic calcification could be made only by fluoroscopy and surgical inspection of the aortic valve, a precise localisation of the calcium deposits could not be made, which might account for the absence of HV prolongation in some patients found to have aortic valvular calcification.

The finding of an increased AH interval in patients with chronic aortic valve disease is more difficult to explain. Four patients with isolated aortic regurgitation had much prolonged AH intervals, measuring more than 190 ms. Two of these patients, one with an AH interval before operation of 390 ms and the other with one of 220 ms, had periods of atrioventricular block after operation; one of these patients had to have a permanent pacemaker implanted.

Cardiac ventricular function in patients with aortic regurgitation was not different from that found in the control group. Moreover, pronounced prolongation of the AH interval was seen both in patients with normal cardiac function and those with a distinct reduction of cardiac index and ejection fraction. Thus, left ventricular dysfunction *per se* does not appear to be an explanation for the observed prolongation of the AH interval. On the other hand, mechanical overstretching of the atrioventricular and interventricular conduction system, resulting from the enlargement of the left ventricle that occurs in aortic regurgitation, might have produced these changes. Study of atrioventricular conduction in other conditions producing diastolic overload of the left ventricle might clarify this possible relation.

The atrioventricular conduction findings in patients with aortic regurgitation produced by endocarditis suggest that the AH prolongation is time-dependent. Patients studied less than 4 months after the occurrence of endocarditis had normal atrioventricular conduction, whereas one patient who was studied 3½ years afterwards showed AH delay. In view of the distinct movement of the aortic root caused by the large stroke volume found in aortic regurgitation and the proximity of the atrioventricular node and proximal His bundle to the aortic root, the AH delay may be the result of injury to these structures caused by such movement over a period of several years. The finding of AH delay in aortic regurgitation is also consistent with a report of complete heart block in these patients (Jensen and Siguard, 1972). Patients with aortic regurgitation usually had atrioventricular junctional escape foci when complete heart block occurred (Jensen and Siguard, 1972), a finding that suggests that the site of block was at the atrioventricular node or in the proximal His bundle. In addition, none of the patients with aortic regurgitation after infective endocarditis had HV prolongation. Though atrioventricular conduction disturbances may occur as a result of direct invasion of the His bundle by a myocardial abscess (Zettner and Irmiere, 1959; Meshel *et al.*, 1970; Kleid *et al.*, 1972), this complication of endocarditis is rare and it is unlikely to have occurred in a group of only 4 patients.

Because cardiac function was not significantly different in the groups studied and patients with aortic valve disease were matched with a group made up largely of patients with coronary artery disease, left ventricular dysfunction or concurrent coronary artery disease could not account for the disturbed atrioventricular conduction found in this study. Failure to identify correctly the His deflection can also be excluded as an explanation for these findings. Particular care was taken to identify the His deflection: (1) deflections occurring 31 ms or less before the V were excluded; (2) recordings were made on the atrial side or in close proximity to the atrium, which was determined by the presence of a large A deflection; (3) multiple deflections were excluded and only characteristic His deflections were used for measurement. Moreover, use of a control group studied in the same manner eliminated inadvertent bias since incorrect identification of the His deflection was as likely to have occurred in both groups.

CLINICAL IMPLICATIONS

The present study suggests another cause of syncope in patients with aortic valve disease. In addition to transient periods of electromechanical dissociation and paroxysmal ventricular tachycardia (Schwartz *et al.*, 1969), intermittent heart block might be another cause. Though patients in the study group were symptomatic, failure to find a relation between haemodynamics and atrioventricular conduction suggests that disturbed atrioventricular conduction might also occur in patients with mild disease or in patients with calcific-sclerotic disease with little or no valvular dysfunction. Thus, Holter monitoring and His bundle electrocardiography should be performed in patients with aortic valve disease who have dizziness and syncope when the haemodynamic disturbance is mild and cannot account for these symptoms.

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